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FUNGUS DISEASE ATTACKING ARTICHOKES.

INCIDENCE, LIFE-HISTORY, AND REMEDIAL TREATMENT.

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DURING recent years the Jerusalem artichoke (*Helianthus tuberosus*) has been widely grown for pig-feed in New Zealand, and the area is increasing each season. The usual method practised by farmers has been to plant the tubers in drills like potatoes, and when the crop has matured to fence off portions and allow pigs to root out and eat the tubers. When the area has been well worked over by pigs these animals are excluded, and the ground is harrowed, permitting a second crop to mature from those tubers overlooked by the pigs. In this manner certain areas have been kept constantly under artichokes for the past four or five years. One disadvantage of this method lies in the fact that as a rule small tubers only are left by the pigs, the yield in consequence becoming less each season. Therefore a modification of this practice is now being advocated by the Department of Agriculture. This consists in lifting, before the pigs are turned on the area, a quantity of large tubers sufficient to plant the area; then when the pigs have been removed the land is cultivated and replanted with these tubers. In this way constancy of crop-yield is secured.

A more serious obstacle to the utilization of the same land for artichokes for several seasons has arisen during the past few seasons, in the form of a disease which has become widespread. Where at all prevalent it has caused the abandonment of the affected areas, as it may at times destroy half or more of the crop. Fresh plantings have been made, and these in turn become infected, usually owing to the use of seed tubers taken from the previously infected crop.

Requests for information concerning this disease and its possible control have become so numerous that the present article—necessarily incomplete—has been written to supply what information is available.

The disease becomes noticeable in a paddock of artichokes in that certain plants in late spring or early summer show signs of wilting, the foliage becomes blackened, and the plant soon is killed. Generally, death of these initial plants (which are almost always overlooked) is followed by wilt and death of numerous others in their vicinity, until definite diseased areas (or "patches," as the farmer terms them) become well established and then readily noticeable. These areas may continue to increase in size gradually through the season, until in the late autumn they may cover an extent of one-third of an acre or even more. Death of the plant is usually followed by its collapse, and the prostrate discoloured stems then become very prominent (Fig. 1).

Examination of a diseased stem shows the presence, usually near ground-level, but often 1 ft. to 2 ft. above the surface of the ground, of a dense white or light-coloured fungous growth covering the stem often for a foot or more of its length (Fig. 2). The tissues lying beneath this region appear dead and discoloured, and embedded in

small enough for a cover to be thrown over them less fumigant will be required and the control will be more effective.

In the vegetable-garden cyanide dust can be effectively used, particularly among cabbages, where large numbers of earwigs are frequently concealed. Apart from its effect on earwigs the cyanide kills other injurious insects, especially aphids.

The writer does not know of any poison that will kill earwigs as quickly as hydrocyanic-acid gas, which proves fatal to them within one to two minutes after application. Caution is necessary in handling this substance; a tin should never be opened in a closed room unless it is particularly well ventilated; every care should also be taken not to inhale the gas.

TRAPS.

Traps for earwigs have been in use for many years, and various types have been employed quite effectively. One such is to place straw in a flower-pot and invert this over a stake driven into the ground. The earwigs seek shelter in the pot during the day, and the contents may then be shaken into hot water or kerosene and the insects destroyed. Pieces of folded sacking or paper left about in tree-crotches or on the ground may similarly be used. Thus by the use of such simple means numbers of the pest may be destroyed.

NATURAL ENEMIES.

Hedgehogs were introduced at Alexandra (Central Otago) in the hope that they would account for a portion of the earwigs, and having in mind also the fact that these animals destroy other pests, notably snails and slugs. Some of these hedgehogs were dissected by Mr. E. F. Northcroft, of the Biological Laboratory, but no earwig-remains were found in the alimentary tracts of the animals. Further dissections will be made in the coming season.

An examination of the excreta of the brown owl (*Athene nocturna*) showed the remains of a large number of earwigs.

CONCLUSIONS.

No definite conclusions can yet be drawn as to which is the best poison to use, since a good deal of field-work still has to be carried out. It is hoped to make a much earlier start on this phase of the work during the coming season. The main advantage of an early application of the poison lies in the fact that insects when young are more readily* poisoned. With regard to traps and barriers, the main feature of the former is perhaps the personal satisfaction obtained, while the latter, though perhaps effective in keeping the earwigs from certain places, does not reduce the numbers. Calcium cyanide as a fumigant is very useful in confined places, but at present is hardly feasible for control in the open.

For assistance in carrying out the field-work here recorded the writer is indebted to Mr. W. R. Lloyd Williams, Orchard Instructor, Alexandra. Thanks are also due to Mr. Kinnaird, at Earnscleugh, for maintaining a constant supply of earwigs during the laboratory experiments.

*Fink, D. E.: Physiological Studies of the Effect of Arsenicals on the Respiratory Metabolism of Insects. *Jour. of Agricultural Research*, vol. 33, pp. 993–1007.

the fungous mycelium appear numerous shot-like, usually black bodies of irregular size and shape, termed sclerotia (Fig. 3). Their significance will be made apparent later.

The wilt and death of plants, their frequent appearance in irregularly circular patches, presence of the fungus growth on the cankered portions of the stem, with accompanying sclerotia, are the characters of the disease.

FIG. 1. DISEASED AREA IN ARTICHOKE FIELD, SHOWING NUMEROUS DEAD, FALLEN STEMS.

[*Photo by Writer.*

HOSTS OF THE CAUSAL ORGANISM.

The disease is by no means confined to artichokes, for in New Zealand it has proved one of the most serious fungous diseases with which we have to contend, attacking as it does so many crop, orchard, and garden plants. The following list of plants on which the causal organism has been collected in New Zealand will give some idea of its wide host range: Antirrhinum (cultivated), artichokes (Jerusalem), bean (broad or Windsor, butter, French, horse, soya), Brassicas (chou moellier, cabbage, kale, rape, swede, and turnip), carnation, Cape gooseberry (*Physalis peruviana*), Canadian thistle (*Cnicus arvensis*), carrot, celery, cucumber, curled dock (*Rumex crispus*), dahlia (cultivated), fat-hen (*Chenopodium album*), fennel (*Foeniculum vulgare*), lettuce, lemon (fruits and shoots), mangold, melon, potato, pea, parsnip, pelargonium (cultivated), passion-fruit, spear thistle (*Cnicus lanceolatus*), sweet-pea, sunflower, spinach, tomato, tree-mallow (*Lavatera arborea*), tree-tomato (*Cyphomandra betacea*), tulip, and wing thistle (*Carduus pycnocephalus*).

The disease has with us in certain seasons proved to be one of the most serious troubles of lettuce, potato, sunflower, and tomato.

CAUSAL ORGANISM.

This disease has been found to be caused by the fungus *Sclerotinia sclerotiorum* (Lib.) Mass. (7), an organism which has been in North America usually named *S. Libertiana* Fcl. As it occurs on many hosts it has at times received many different names, those of recent application being *S. intermedia* Ramsey (9), *S. minor* Jagger (5), and *S. perplexa* Lawrence (6); for comparisons of the published descriptions of these show they all agree with *S. sclerotiorum*, differing only in certain characters which cannot be considered as possessing specific value.

FIG. 2. DISEASED PLANT, SHOWING WHITE FUNGOUS GROWTH ON STEMS. REDUCED.

[*Photo by Writer.*

Identification of the fungus on the hosts enumerated was carried out in two ways: (1) The sclerotia were germinated and ascospores obtained; (2) sclerotia were plated to media and their cultural characters studied. These were compared with cultures made from ascospores, and all were found to agree to the extent that separation of any one was not possible, although considerable variation was noted in certain particulars, such as growth-rate of the mycelium, size of sclerotia produced on media, &c. Apothecia were obtained from germinated sclerotia taken from artichoke, carrot, celery, cucumber, lettuce, pea, potato, tomato, and tree-mallow.

LIFE-HISTORY.

Opinions differ among pathologists as to the method by which infection occurs in the field, one view being that ascospores produced from germinating sclerotia in the spring cannot attack the host directly, but must first develop a copious saprophytic mycelium (presumably in the soil) which later attains the parasitic condition. This statement is invariably attributed to de Bary, but, as Pethybridge has pointed out (see below), his original statement did not convey this meaning. This opinion was regularly held by most of those who have published on this organism, until a paper by Pethybridge appeared (8), in which direct infection by ascospores of potato-haulms was secured. His observations are so informative as to bear reproduction:—

> In the spring and summer these sclerotia, which are then to be found in and on the soil, germinate and produce stalked "spore-cups" (apothecia), from which little puffs or clouds of spores can be seen to proceed. Nothing could be simpler than to suppose that the potato-plants become infected directly from these air-borne spores. Arguments have, however, been brought forward which seem to show the impossibility, or at least the improbability, of this mode of infection. Thus de Bary found that when the spores germinated in water the germ tubes were

quite unable to penetrate the living healthy tissue of the plant. If, however, germination took place in a nutritive solution, instead of in water only, the germ tubes were found to be capable of producing infection. So impressed was de Bary with this difference in behaviour of the spores according as to whether they germinated in pure water or in a food solution that he went so far as to state (3, p. 380) that the fungus always requires to pass through a previous stage of existence as a saprophyte in order to be capable of parasitism. The expression "previous stage of existence" appears to have had a somewhat exaggerated importance attached to it, at least from the practical point of view, and has led to the idea being promulgated that in one stage of its life-history the fungus lives saprophytically—*i.e.*, in decaying manure or organic matter in the soil—and hence it has generally been maintained that the potato-plant becomes attacked at soil-level by the invasion of saprophytically nourished mycelium from the soil. This view of the mode of infection by this fungus is, of course, supported by the experiments carried out by de Bary on potted plants of zinnia (2, p. 378). Ten of such plants were contained in a single pot of soil, and placed in contact with the base of the stem of one of them was a piece of carrot on which the fungus was living. Ultimately nine out of the ten plants succumbed, the attack in each case taking place at the soil-level. This result can scarcely be regarded as surprising, but it is not necessary to conclude from it that in the field the attack must always come from the soil . . . Up to the present, at any rate, no one has reported the finding of this fungus living naturally in the soil as a saprophyte.

It is true that in the case of the potato the point of attack is often at or near soil-level . . . But . . . the points of attack are very frequently found a considerable distance above soil-level, so that there can be no question of infection from the soil at such places.

In the same paper Pethybridge records experiments in which he proved conclusively that haulms and leaves could become infected directly by ascospores. He further remarks (p. 17) :—

Observations of plants in the plots strongly suggest that in the main an entry into the plant is gained in two or three ways. The first of these is through the older, shaded, yellowing leaves, which ultimately fall off. The fungus is not infrequently found on them, and makes its way down the leaf-stalk, and thus its mycelium reaches the stem of the plant and enters it. It seems quite possible that the germinating spores might be able to secure an entry into such dying leaves, whereas they would still be unable to directly infect perfectly healthy ones . . . The second point of entry which suggests itself consists of the series of wounds left on the stalks by the falling leaves. In any case it is a fact that far the greater number of cases of infection occur at a node . . . The essential fact which has been brought out by those experiments of the past season remains —viz., that the attack of the potato-plant with this disease takes place chiefly from aerially-borne spores, and not, as has hitherto been maintained, from the soil.

Dowson (4), working with this organism on antirrhinums, was able to produce infection when ascospores were either inoculated into wounds or placed directly on the host-tissues. He also succeeded in obtaining infection with mycelium. Ramsey (10) was able to produce severe infection by spraying a suspension of ascospores over the freshly cut leaves of lettuce. Thus recent workers have shown ascospore infection of the host occurs quite readily.

Field observations on artichokes show that infection some distance above the ground is quite common ; and from what has been written it is obvious that such infection must occur from ascospores. Consequently, despite the observations of earlier workers, ascospore infection must be considered as the source of primary infection. This view is strengthened by the fact that these early infections occur in late spring or early summer, the period when apothecia are readily obtained from germinating sclerotia. Sclerotia, as has been shown, are black, shot-like bodies produced in numbers on the cankered

portions of the stem. When mature they fall to the ground, where they usually remain quiescent until the following spring. They are in reality compact bodies composed of intricately interwoven hyphæ. From them arise under favourable conditions (in the spring, during periods when abundant moisture is present, though they are not uncommonly produced through the summer months) stalked, saucer-shaped, light-coloured structures termed apothecia. These bear on their upper surfaces numerous cylindrical sacs, each containing eight spores. These are discharged forcibly into the air and are carried to plants in the vicinity. Here, conditions being favourable, infection occurs, and a mycelium is produced, which penetrates the plant-tissues, killing them and blocking the conduction-vessels. As a result, wilt and death of the plant follows, the mycelium at the same time growing to the surface of the attacked portion, where it produces sclerotia, which eventually fall to the ground.

FIG. 3. SHOWING BLACK SCLEROTIA ON STEMS. HALF NATURAL SIZE.
[Photo by Writer.

The disease frequently does not spread beyond these initially infected plants, its spread being dependent to a large extent on a high moisture content of the soil and vicinity. Usually, however, it spreads to contiguous plants, forming a small diseased patch; or it may spread rapidly over a considerable area, until in extreme cases all plants within an area of one-third of an acre may be killed. The manner of the spread of the disease from the initially to subsequently infected plants is not known, but, judging from the number of stems infected well above ground-level, it is probable that here, too, ascospore infection is chiefly responsible.

Remedial Treatment.

Inspection of diseased plants shows that partial or complete infection of the tubers is frequent—this, of course, depending on the period at which infection occurs. When tuber-infection is severe most of them rot, sclerotia being produced in abundance on the exterior of decayed

tubers. Others may show slight infection, which often escapes notice, and it is these tubers, often with one or several sclerotia attached (frequently inevident, as they are in the bases of the buds and are usually covered with earth), that carry the disease to clean land. As has been shown, once a crop becomes infected the disease persists in that crop, being carried over from season to season by the sclerotia remaining in the soil. On clean land—land out of grass—such bodies are not present; therefore it is obvious that the disease must be carried to the ground on the seed tubers when it appears in artichokes growing therein.

This knowledge enables us to prevent the disease becoming a serious trouble to those desirous of growing artichokes for pig-feed. But, unfortunately, nothing can be done to prevent infection in those areas where artichokes are already established and in which the disease is present. Therefore, when the disease is severe such areas should be abandoned and sown to grass; for when the host range of this fungus is considered it will be seen that it is unsafe for the farmer to sow on infected land for at least two seasons such crops as mangolds, peas, potatoes, &c.

When it had been determined that the disease was carried in the tuber the writer carried out numerous experiments with a view to ascertaining whether the organism could be destroyed without detriment to the tuber. Tubers were washed, and dipped for varying periods of time in mercuric-chloride solutions according to formulas published elsewhere (1) for the control of corticium-disease of potatoes. They were then planted in boxes. Results showed that artichoke tubers would not tolerate immersion in these solutions, those dipped for five minutes only in 1–1,000 $HgCl_2$ rotting in fifteen days after treatment. Moreover, at this strength the fungus was not killed, growth being readily obtained from sclerotia plated to media from the dipped tubers.

Further, it was found that the disease often was present in mycelial form within the tubers, being present in discoloured tissue at the ends by which the tubers were attached to the parent plant. Penetration of a fungicide through this tissue is not possible, so that, even were a solution evolved capable of killing the sclerotia on the exterior, the disease would still be carried in this diseased tissue. It was found necessary before treatment to wash tubers free from all attached earth, and it was during this operation that the stem-end diseased areas containing mycelium (and often sclerotia) of the pathogen were noted.

Thus remedial measures would consist in using disease-free tubers for planting. To obtain these it would be necessary to take seed tubers from a field in which the disease apparently was absent, to wash these free from soil, and to discard *any* showing discoloured spots. In addition it would be necessary to plant on land which had not been under swedes, turnips, mangolds, vegetables, peas, beans, garden flowers, &c., for at least two seasons previously. In fact, it would be advisable to use for the purpose ground which had been in grass for some two or three years. Although the disease has been found on such weeds as curled dock, spear and wing thistles, fat-hen, and Canadian thistle, its occurrence on these hosts in nature is so rare that

danger of infection from these sources may be overlooked, especially where lea land is to be used for planting.

Finally, farmers are warned against feeding to pigs on land in artichokes (or to be used for this purpose) diseased bulbs of swede and turnip, garden vegetables, or the like, or feeding such to pigs in pens and dumping the manure from the pens on land to be used for artichokes, for it is in this manner that infection frequently occurs.

A second possible method of dealing with this trouble has recently been brought to the writer's notice by Mr. N. R. Foy, Seed Analyst. While on a recent visit to Taranaki Mr. Foy found that certain farmers in the Eltham district were growing the pink instead of the usual white variety of artichoke, for they claimed that this variety was highly resistant to the disease. The pink variety is not such a prolific cropper as the white; nevertheless, if resistant, it is worth consideration, for it could then be more safely planted on the same land on which whites had been severely infected.

The writer is indebted to Mr. K. W. Gorringe, Instructor in Swine Husbandry, Department of Agriculture, Wellington, for particulars regarding the methods used in growing and feeding off artichokes; to Mr. N. R. Foy, Seed Analyst of this Laboratory, for particulars concerning the use of artichoke varieties in Taranaki; and to Mr. A. C. Buist, New Zealand Farmers' Co-operative Association, Ltd., Feilding, for conducting him to diseased areas in that district, procuration of specimens, and the like.

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Agricultural Legislation.—Drafts of the following Bills have been recently considered and approved by the Board of Agriculture: Fertilizers, Importation of Plants, Staining of Imported Seed, Stock Amendment, and Noxious Weeds Amendment.